Muscle Function Following Post-Stroke Locomotor Training: A Simulation Analysis of Different Strategies to Improve Walking Speed.

by

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Abstract

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The assessment of rehabilitation effectiveness in the post-stroke hemiparetic population has primarily focused on walking speed. Walking speed, however, may be improved through a number of mechanisms; increased speed can be achieved through a combination of increased propulsion (propelling the center of mass forward) and swing initiation (resulting in longer and faster steps) in either the paretic or nonparetic leg. Therefore the objective of this study was to use a detailed musculoskeletal model and forward dynamics simulations to identify the individual muscle contributions to forward propulsion and swing initiation following locomotor training in two post-stroke hemiparetic patients who had similar speed increases following training, one utilizing an "ankle strategy" (increases in ankle power generation to accelerate the trunk forward) and

one a “hip strategy” (increases in hip flexor generation of the swing leg to accelerate the leg forward) to increase speed. Each subject participated in locomotor therapy training using a body weight supported treadmill modality. Strategy classification was based on inverse dynamics analysis pre- and post-training. The simulation analyses revealed that forward propulsion was achieved primarily through the uniarticular plantarflexors and the contralateral knee extensors in both subjects. The main difference between the two strategies occurred primarily in the hip muscle contributions to swing initiation. The “hip strategy” subject, in addition to using the hip flexors to accelerate the leg forward, had higher contributions from the contralateral non-sagittal plane hip muscles to generate energy to the leg to initiate swing. These results suggest that using either the “ankle strategy” or the “hip strategy” to increase speed post-training results in similar muscle function post-training walking with differences primarily occurring in the hip muscle contributions to swing initiation. Future studies analyzing both pre- and post-training may reveal changes in muscle function that correspond more with the strategy classifications.

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Chapter One

INTRODUCTION

The assessment of rehabilitation effectiveness in the post-stroke hemiparetic population has predominantly focused on walking speed since walking speed is related to functional status and quality of life (Bowden et al., 2008; Perry et al., 1995). However, impairments in walking speed can occur through a number of mechanisms. For example, increased speed can be achieved through a combination of increased propulsion (propelling the body center of mass forward) and swing initiation (resulting in longer and faster steps) in either the paretic or nonparetic leg. The contributions of individual muscles to these subtasks in healthy walking and how they change with increased speed are well understood (Liu et al., 2006; Neptune et al., 2001; Neptune et al., 2008; Neptune et al., 2004); the ankle plantarflexors are the primary contributors to forward propulsion (Liu et al., 2006; Neptune et al., 2001) while the hip flexors and biarticular plantarflexors are the primary contributors to swing initiation (Neptune et al., 2004). However, individual muscle contributions to these walking subtasks in the post-stroke population and the effects of improved walking speed post locomotor rehabilitation training are not well understood. Identifying individual muscle contributions is essential to design effective rehabilitation strategies.

Current literature suggests that speed increases following locomotor training usually occur using one of two strategies, an “ankle strategy” or a “hip strategy” (Lewis and Ferris, 2008; Mueller et al., 1994). The “ankle strategy” increases ankle power generation to increase ankle push-off, thus propelling the leg into swing and accelerating the trunk forward, while the “hip strategy” uses the hip flexor muscles of the swing leg to accelerate the leg forward (Mueller et al., 1994). The “ankle strategy” is normally used

to increase speed in healthy walking (Neptune et al., 2008), however, many populations exhibit plantarflexor weakness and are limited in the amount of power that can be generated at the ankle. These populations (e.g. elderly, diabetes, transtibial amputees) often utilize the "hip strategy" to compensate for weakened ankle plantarflexors (Judge et al., 1996; Mueller et al., 1994; Sadeghi et al., 2001; Zmitrewicz et al., 2007). In the stroke population there is much variability, with some individuals exhibiting reduced plantarflexor and others reduced hip flexor strength compared to healthy individuals (Chen and Patten, 2008; Lamontagne et al., 2007; Olney and Richards, 1996). Thus the "ankle strategy" and "hip strategy" are both used to increase speed in post-stroke hemiparetic patients with the strategy chosen most likely dependent on the neuromuscular resources available to them (Jonsdottir et al., 2009; Nadeau et al., 1999; Parvataneni et al., 2007).

While many studies have examined post-stroke walking related to speed, most have focused on the change in walking speed from self-selected to fastest-comfortable walking measured on the same day (Chen et al., 2005; Jonkers et al., 2009; Kim and Eng, 2004; Milot et al., 2007; Nadeau et al., 1999; Olney et al., 1994) rather than following training (Parvataneni et al., 2007; Sullivan et al., 2007; Teixeira-Salmela et al., 2001). In addition, these studies have focused on net joint quantities that are unable to elucidate the contributions of individual muscles to walking subtasks. Muscle driven forward dynamics simulations are ideal for investigating individual muscle function and have been successfully used to study walking in both healthy (Liu et al., 2006; McGowan et al., 2008; Neptune et al., 2001; Neptune et al., 2004) and a variety of patient populations (Goldberg and Neptune, 2007; Higginson et al., 2006; Zmitrewicz et al., 2007).

Therefore the objective of this study was to use a detailed musculoskeletal model and forward dynamics simulations to identify the individual muscle contributions to the

walking subtasks of forward propulsion and swing initiation following locomotor training in two post-stroke hemiparetic patients who had similar speed increases following training, one utilizing the "ankle strategy" and one the "hip strategy" to increase speed. Understanding the individual muscle contributions to the walking subtasks in these two subjects post-training will provide insight into the effectiveness of the different neuromuscular control strategies used by post-stroke hemiparetic subjects to increase speed following training.

METHODS

Subjects

The subjects for this study were selected out of a larger pool of subjects with chronic hemiparesis: one that exemplified the "ankle strategy" (F, 44 y.o., right side paretic, 8 months since stroke) and one the "hip strategy" (M, 55 y.o., left side paretic, 12 months since stroke). Subjects participated in a 12-week locomotor rehabilitation training program designed to improve walking speed and mechanics. The inclusion criteria were hemiparesis secondary to a single unilateral stroke; no significant lower extremity joint pain, range of motion limitations, or major sensory deficits; able to ambulate independently with an assistive device over ten meters on a level surface; able to walk on a daily basis in the home; no severe perceptual or cognitive deficits; no significant lower limb contractures; and no significant cardiovascular impairments contraindicative to walking. All subjects passed an exercise tolerance test (Yates et al., 2004) to verify their cardiovascular fitness for locomotor training therapy before beginning training.

Training

The training program was a 12-week locomotor training therapy three times a week with each session containing 20 minutes of actual stepping using a body weight supported treadmill modality (Hesse et al., 1995; Plummer et al., 2007; Visintin et al., 1998) followed by 20 minutes of immediate translation of skills acquired during walking on the treadmill to overground walking. Training began with 40% body weight support (BWS) and progressed as tolerated across sessions to no BWS. Training took place between 2.0 and 3.0 mph with manual assistance provided by physical therapists at the hip and/or lower legs to approximate trunk, pelvis, and limb kinematics as well as the spatial-temporal pattern of walking (Plummer et al., 2007).

Experimental Protocol

Subjects performed a 30-sec walking trial on a split-belt instrumented treadmill (Techmachine, Andrézieux Boutheon, France) at their fastest comfortable speed both pre- and post-training. A safety harness mounted to the laboratory ceiling was worn across the shoulders and chest to protect the subject in the event of loss of balance. One or more practice trials were performed to ensure subjects were comfortable with the setup. Subjects walked approximately 10 seconds prior to each data collection to ensure a steady-state walking pattern had been reached. A modified Helen Hayes reflective marker set was recorded using an twelve-camera motion analysis system (Vicon Motion Systems) to capture bilateral 3D kinematics at 100 Hz. A 16-channel EMG system (Konigsburg Instruments, Pasadena, CA) was used to record bilaterally at 2000 Hz from the medial gastrocnemius, soleus, tibialis anterior, rectus femoris, vastus lateralis, biceps femoris, semimembranosus, and gluteus medius. Bilateral 3D ground reaction forces, moments, and center of pressure were collected at 2000 Hz.

Data Processing and Analysis

Visual3D (C-Motion, Inc., Germantown, MD) was used to process all data. Marker and ground reaction forces (GRFs) were low pass filtered using a 4th order Butterworth filter with cutoff frequencies of 6 Hz and 20 Hz, respectively. EMG was high pass filtered with a cutoff frequency of 40 Hz, de-meaned and then low pass filtered with a cutoff frequency of 10 Hz using a 4th order Butterworth filter. A standard inverse dynamics analysis was performed to calculate 3D joint moments and powers. An inverse kinematics analysis was then performed to find the model joint angles that best reproduced experimental body kinematics.

To analyze the data, each stance phase was subdivided into event defined regions based on GRFs (Fig 1). Region 1 corresponded to the first double support phase following heel strike, region 2 corresponded to the first 50% of single leg stance, region 3 corresponded to the second 50% of single leg stance, and region 4 corresponded to the second double support during pre-swing (Turns et al., 2007). To classify each subject as using a "hip strategy" or an "ankle strategy", the joint moment impulses pre- and post-training were used. Hip (flexor positive) and ankle (dorsiflexor positive) joint moment impulses (time integral of the corresponding joint moment) were calculated during both paretic and nonparetic regions 3 and 4, which approximately correspond to the propulsive phase.

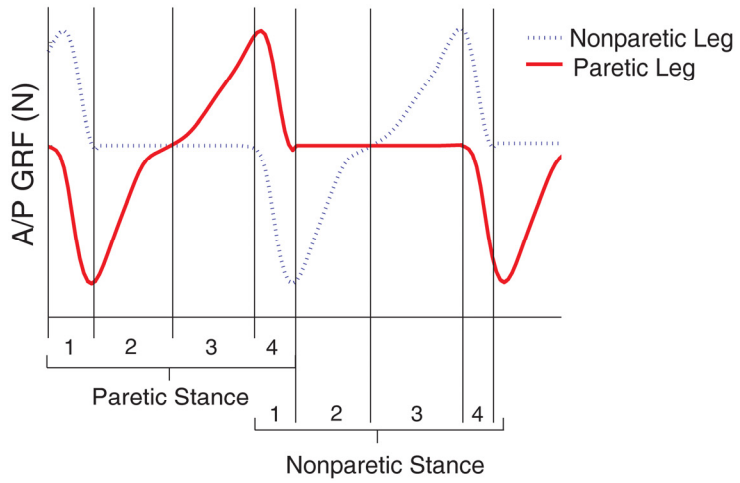

Figure 1: Paretic and nonparetic stance phase regions

Musculoskeletal model

A 3D forward dynamics musculoskeletal model and simulation were developed using SIMM (MusculoGraphics, Inc., Santa Rosa, CA) based on a previously developed 2D model (Neptune et al., 2001). The model consisted of rigid-body segments representing the trunk, pelvis and legs. Each leg consisted of thigh, shank, patella, foot and toe segments. The musculoskeletal geometry was based on Delp et al (1990) and the trunk segment included the mass and inertial characteristics of the head, arms, and thorax. The pelvis was free to translate and rotate with respect to the ground with six degrees-of-freedom (three translational, three rotational). The trunk had three rotational degrees of freedom with respect to the pelvis. Each hip and knee joint was modeled using a spherical and revolute joint, respectively. The patella was constrained to move along a prescribed trajectory relative to the femur as a function of knee flexion angle to assure an appropriate moment arm for the muscles crossing the knee joint (Yamaguchi and Zajac, 1989). The ankle, subtalar, and metatarsal-phalangeal joints were modeled as revolute joints. Thus, the model had a total of 23 degrees-of-freedom. Passive torques were applied at the hip, knee, ankle, and subtalar joints to represent forces applied by

ligaments and other passive structures (Davy and Audu, 1987), the metatarsal-phalangeal joint was controlled by a passive-visco elastic torsional spring that represents muscles, tendons and ligaments spanning the joint that were not included in the model, and the pelvis-trunk joint was controlled by three passive visco-elastic torsional springs that represent the abdominal and lower back muscles. Contact between the foot and ground was modeled using 31 visco-elastic elements with coulomb friction distributed across the bottom of each foot segment (Neptune et al., 2000).

The system dynamical equations of motion were generated using SD/FAST (PTC, Needham, MA) and a muscle-actuated forward dynamics simulation was produced using Dynamics Pipeline (MusculoGraphics, Inc, Santa Rosa, CA). The model was driven by 38 individual Hill-type musculotendon actuators for each leg that were combined into 34 muscle groups based on anatomical classification (Table 1), with muscles within each group receiving the same excitation pattern. Each group was excited by either one or two excitation blocks (Table 1) based on subject specific EMG. For those muscles that EMG was not available previous experimental data were used. (Perry, 1992; Sutherland, 2001) Each excitation block pattern was defined by excitation onset, duration and magnitude. Contractile dynamics were governed by Hill-type muscle properties including the force–length–velocity relationships (Zajac and Gordon, 1989). A first order differential equation was used to represent muscle excitation-activation dynamics (Raasch et al., 1997), with activation and deactivation time constants derived from Winters and Stark (1988). For those muscles that data were not explicitly available, nominal values of 12 and 48 ms were used.

Dynamic optimization

Dynamic optimization was used to generate a walking simulation for each subject from paretic mid-stance to non-paretic toe off in order to capture both double support phases of the gait cycle. The optimal tracking solution was solved using a simulated annealing optimization algorithm (Goffe et al., 1994). The onset, duration and magnitude of each excitation burst and the initial joint angular velocities were optimized to minimize the difference between the simulated and experimentally measured data (290 and 263 combined total parameters for both the paretic and nonparetic leg, for Subject 1 and 2, respectively). The experimental kinematics were taken from the single gait cycle whose kinematics best represented the average kinematics over the entire 30s trial. The objective function in the optimization minimized the squared error normalized by the inter-trial variability for each quantity tracked (Neptune et al., 2001) in the following form:

$$J = \sum_{j=1}^{m} \sum_{i=1}^{n} \frac{\left(Y_{ij} - \hat{Y}_{ij}\right)^2}{SD_j^2}$$

where Y_{ij} = measurement of variable j at time step I, $\hat{Y}_{ij}$ = simulation data corresponding to Y_{ij}, SD_j^2 = average inter-trial variability of variable j. The quantities evaluated included: 3D pelvis angles and translation, 3D trunk angles, 3D hip angles, sagittal plane knee and ankle angles, and the GRFs.

Muscle function analysis

To identify how each subject increased their walking speed following locomotor training, muscle-induced acceleration and segmental power analyses were performed to quantify the individual muscle contributions to forward propulsion and swing initiation

post-training (Fregly and Zajac, 1996; Neptune et al., 2001; Neptune et al., 2004). A muscle's contribution to forward propulsion was determined by the average power generated in the horizontal direction to the trunk and pelvis during regions three and four. Paretic and nonparetic propulsion were defined as forward propulsion during paretic and nonparetic regions three and four, respectively. A muscle's contribution to swing initiation was determined by first quantifying the power delivered to the leg segments at each instant and then integrating the power over pre-swing (region 4) to find the energy delivered to the leg. Paretic and nonparetic swing initiation was defined as swing initiation during paretic and nonparetic region 4, respectively. Muscles on each leg were combined into 18 groups based on similar anatomical and functional classification for the functional analysis (Table 1).

Table 1: The 38 muscles on each leg were combined into 34 groups, with muscles within each group receiving the same excitation, and then into 18 groups when analyzing muscle function. Excitation patterns for each group consisted of one or two blocks (paretic, nonparetic when two values listed).

Muscle name	Analysis Group	Excitation group	# Excitation Blocks Subject 1	Subject 2
Iliacus	IL	IL	1	1
Psoas	IL	IL	1	1
Adductor Longus	AL	AL	1	1
Adductor Brevus	AL	AB	1	1
Pectineus	AL	PECT	1	1
Quadratis Femoris	QF	QF	1	1
Adductor Magnus 1	AM	AM1	1	1
Adductor Magnus 2	AM	AM2	1	1
Adductor Magnus 3	AM	AM3	1	1
Sartorius	SAR	SAR	1	1
Rectus Femoris	RF	RF	2	2
Vastus Medialis	VAS	MVAS	1	1
Vastus Lateralis	VAS	LVAS	1	1
Vastus Intermedialis	VAS	LVAS	1	1
Gluteus Medius 1	AGMED	GMED1	2	2,1
Gluteus Minimus 1	AGMED	GMIN1	2	2,1
Gluteus Minimus 2	AGMED	GMIN2	2	2,1
Gluteus Minimus 3	AGMED	GMIN3	2	2,1
Gluteus Medius 2	PGMED	GMED2	2	2,1
Gluteus Medius 3	PGMED	GMED3	2	2,1
Piriformis	PGMED	PIRI	2	2,1
Gemellus	GEM	GEM	2	2,1
Tensor Fascia Lata	TFL	TFL	1	1
Gluteus Maximus 1	AGMAX	GMAX1	1	1
Gluteus Maximus 2	AGMAX	GMAX2	1	1
Gluteus Maximus 3	PGMAX	GMAX3	1	1
Semitendinosus	HAM	MH	1,2	1,2
Semimembranosus	HAM	MH	1,2	1,2
Gracilis	HAM	GRAC	1,2	1,2
Biceps Femoris Long Head	HAM	BFLH	1,2	1
Biceps Femoris Short Head	BFSH	BFSH	1	1
Medial Gastrocnemius	GAS	MGAS	1	1
Lateral Gastrocnemius	GAS	LGAS	1	1
Soleus	SOL	SOL	1	1
Tibialis Posterior	SOL	TP	1	1
Flexor Digitorum Longus	SOL	FDL	1	1
Tibialis Anterior	TA	TA	1	1
Extensor Digitorum Longus	TA	TA	1	1

RESULTS

Subject classification

Following locomotor training, Subject 1 walked at 1.0 m/s and Subject 2 at 1.1 m/s, which corresponded to an increase in speed of 0.30 m/s for both subjects. Subject 1 pre-training had very little contribution from the paretic leg to forward propulsion (Table 2). Following training, the contribution to propulsion from paretic leg increased, presumably resulting in the observed increase in walking speed. This was achieved through increased ankle plantarflexor output (summed increases in the plantar flexor moment impulse during region 3 and 4 of 10.5 paretic and 6.9 nonparetic, Nm•ms, %BW, Fig. 2). Subject 2 initially had symmetric propulsion generation between legs (Table 2). The primary mechanism for increased speed was hip flexor output from both legs (increases in the hip flexor moment impulse 8.2 paretic and 4.5 nonparetic, Nm•ms, %BW, Fig. 2) while there was only a small change in paretic ankle plantar flexor output (1.6 Nm•ms in region 4, %BW, Fig. 2). The largest increases in the hip flexor output occurred in region 4, the double support phase preceding swing, and thus likely contributed to swing initiation. Based on these results, Subject 1 was considered to use the "ankle strategy" and Subject 2 the "hip strategy" to increase speed.

Table 2: Propulsion generated by each leg pre- and post-training.

	Paretic Propulsion (Ns,%BW)		**Nonparetic Propulsion (Ns,%BW)**	
Subject	**Pre**	**Post**	**Pre**	**Post**
1	4.3	33.3	70.3	67.4
2	29.4	37.3	31.6	47.6

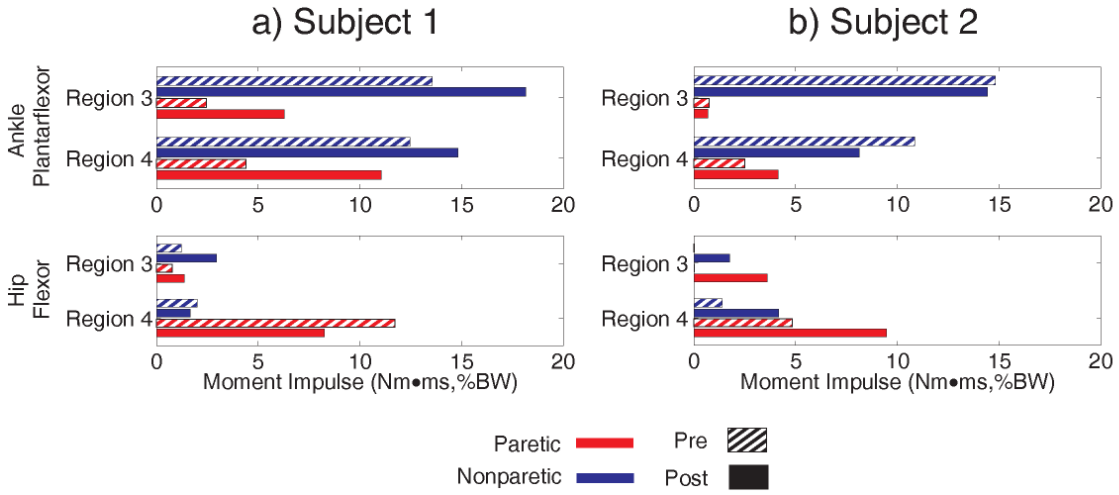

Figure 2: Hip flexor and ankle plantar flexor moment impulses pre- and post-training.

Simulation tracking

The simulations emulated well the experimental data for both subjects (Fig. 3) with few exceptions. Larger deviations were seen at the beginning of the simulation due to the guesses for the initial conditions; however the analysis focused on the second half of the stance phase when the tracking improved. In addition, there were consistent deviations in ankle angle tracking. We were interested in accurately tracking the ground reaction forces, therefore they were weighted high in the multi-objective cost function resulting in less accurate ankle angle tracking. There were also deviations in the nonparetic hip adduction and knee flexion tracking in Subject 1 during paretic preswing that may have a small effect on the results. Finally, the muscle excitation timing corresponded well with the experimentally recorded EMG (see Appendix).

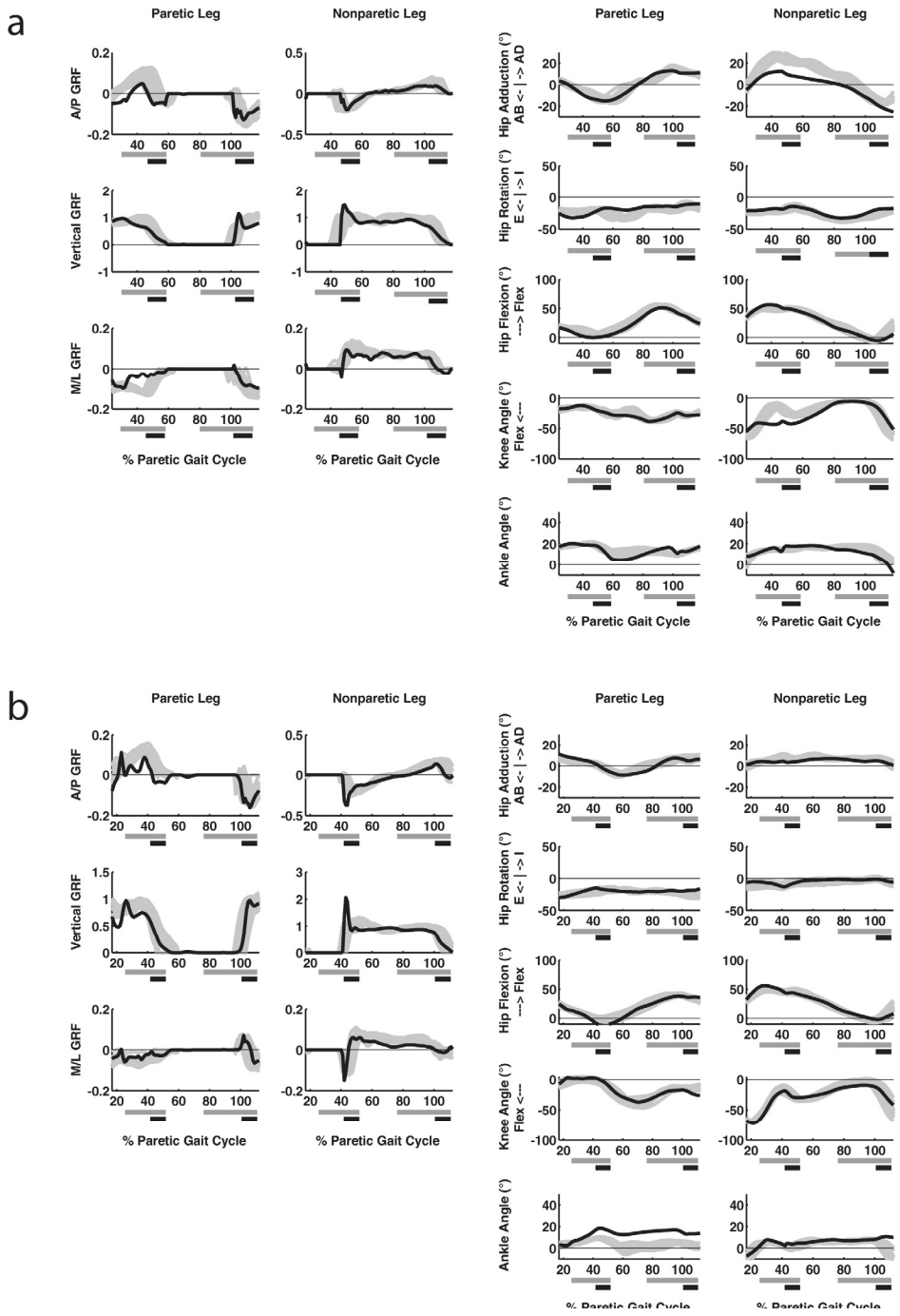

Figure 3: Simulation tracking results (black lines) for (a) Subject 1 and (b) Subject 2 show close agreement with the experimental data (gray lines). GRFs are normalized to body weight. Gray lines represent individual step cycles. Gray and black bars indicate the propulsive phase and pre-swing, respectively.

Muscle function

Subject 1 ("ankle strategy")

Muscle contributions to propulsion (positive horizontal power to trunk and pelvis) by Subject 1 during the paretic propulsion phase (Fig. 4a) were high, with contributions from the nonparetic RF, paretic VAS and paretic SOL being the primary contributors. During the nonparetic propulsive phase (Fig. 4b) the nonparetic SOL, paretic VAS, and nonparetic GAS were the top contributors. Paretic swing initiation in Subject 1 was achieved primarily through contributions from the paretic AL and nonparetic GMED (Fig 4c) while the principal muscles contributing to nonparetic swing initiation were the nonparetic HAM and IL as well as the paretic GMED and AM (Fig 4d).

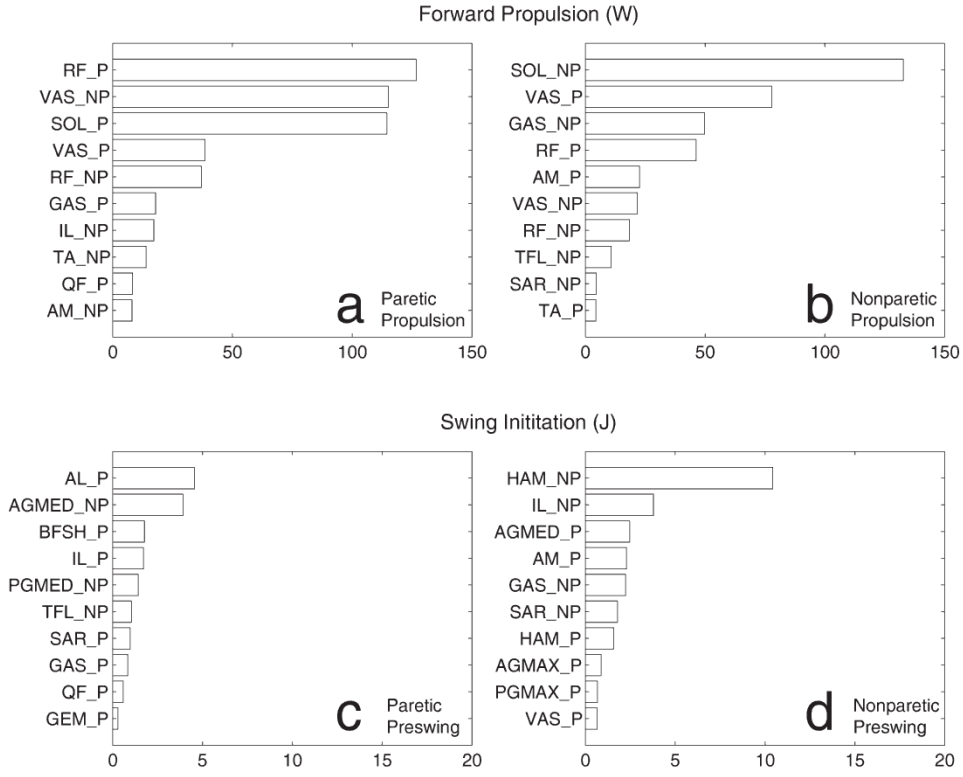

Figure 4: Subject 1 muscle contributions to forward propulsion and swing initiation

Subject 2 ("hip strategy")

The primary contributors to propulsion for Subject 2 during paretic propulsion phase (Fig. 5a) were the paretic SOL and nonparetic VAS. During the nonparetic propulsion phase (Fig 5b) the primary contributors were the nonparetic SOL and paretic VAS. The primary contributors to paretic swing initiation in Subject 2 (Fig 5c) were the nonparetic anterior GMED and HAM, and the paretic QF, AL and IL. The primary contributors to nonparetic swing initiation (Fig 5d) were the nonparetic HAM and GAS and paretic AM and HAM.

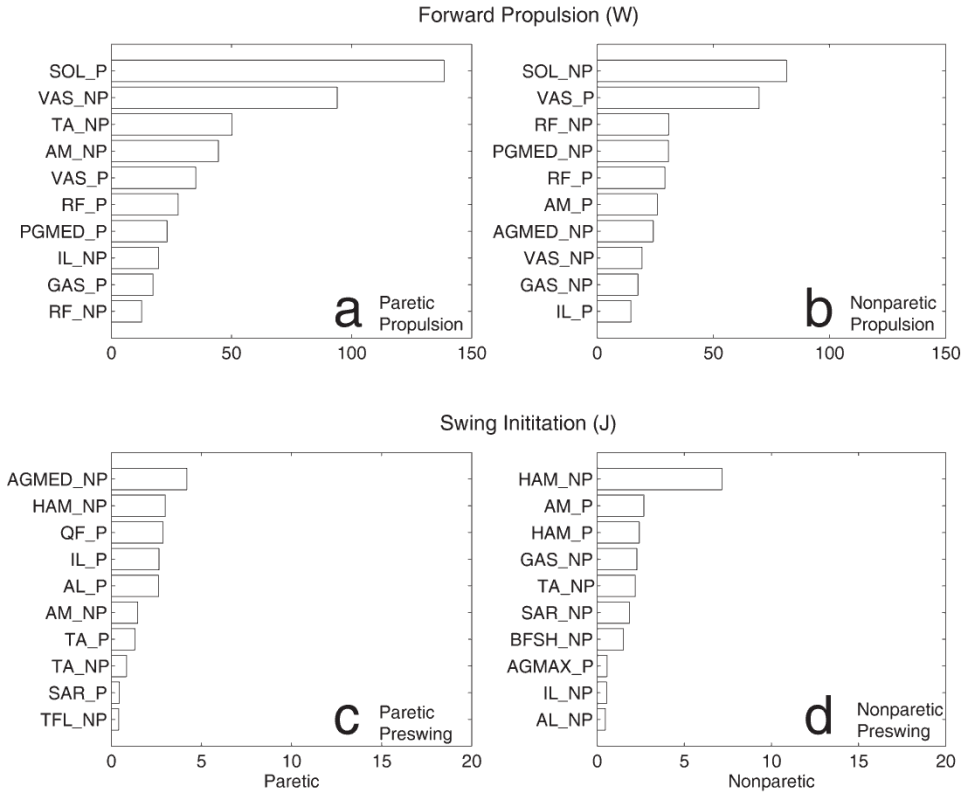

Figure 5: Subject 2 muscle contributions to forward propulsion and swing initiation

DISCUSSION

The goal of this study was to examine individual muscle function in two post-stroke patients who utilized different compensatory strategies (ankle or hip strategy) to increase speed following a locomotor training program. Strategy classification was based on inverse dynamics analyses of experimental data pre- and post-training and a muscle driven forward dynamics analysis was performed for each subject post-training to identify the individual muscle contributions to forward propulsion and swing initiation. Post-training muscle function was similar in both subjects and the main difference between strategies was the level of contributions from individual muscles to the walking subtasks.

Forward propulsion was achieved primarily through the uniarticular plantarflexors and the contralateral knee extensors in both subjects. The "ankle strategy" subject had higher contributions from the plantarflexors in the nonparetic leg; however, the contributions in the paretic leg were similar between both subjects. The main difference between subjects occurred in swing initiation, which was generated by both the ipsilateral and contralateral hip muscles. The "ankle strategy" subject relied more on the ipsilateral hip flexors while the "hip strategy" subject relied more on the contralateral non-sagittal plane hip muscles.

Forward Propulsion

Similar to previous studies, the ipsilateral ankle plantarflexors contributed to forward propulsion (Liu et al., 2006; McGowan et al., 2008; Neptune et al., 2001; Neptune et al., 2008); both SOL and GAS contributed to forward propulsion, with higher contributions from SOL. In the present study the contralateral knee extensors were also significant contributors to forward propulsion. The contralateral VAS was among the

primary contributors for both subjects with contributions at times equal to or greater than the ipsilateral plantarflexors. This contribution was not observed in previous simulation studies of healthy walking (Liu et al., 2006; Neptune et al., 2004). However, these studies defined forward propulsion using measures representing the net effect of the leg and trunk (i.e., AP GRFs and total body COM). Here we chose to represent forward propulsion using positive horizontal power delivered to the trunk and pelvis in order to separate the muscle contributions to individual segments (e.g. accelerating the leg forward versus accelerating the trunk forward). We found the contralateral VAS contributes to forward propulsion through a combination of pelvis rotations. During the ipsilateral propulsive phase, the contralateral VAS rotated the pelvis in the sagittal and transverse planes such that the pelvis and trunk center of mass (COM) were accelerated forward, thus contributing to forward propulsion in both subjects (Fig. 6a). In addition, the paretic RF was an important contributor to forward propulsion, transferring power from the legs to the pelvis and trunk during both the paretic and nonparetic propulsive phases (Fig 7). These contributions were higher in the "ankle strategy" subject.

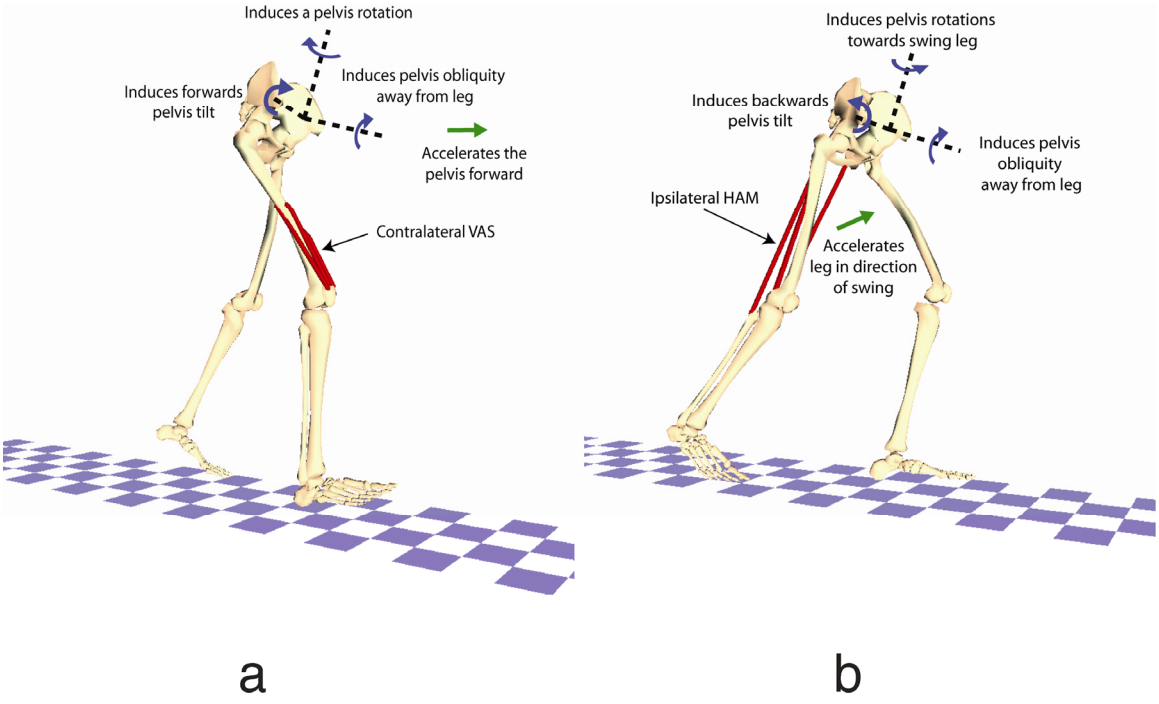

Figure 6: (a) The contralateral VAS, during the ipsilateral propulsive phase, induced forward acceleration of the pelvis. The contralateral VAS rotates the pelvis and tilts the pelvis forward such that the pelvis COM is accelerated forward. (b) The ipsilateral HAM, during pre-swing, contributes to swing initiation. The HAM tilts the pelvis backwards and rotates the pelvis toward the swing leg, both accelerating the leg COM forward. In addition, a pelvis obliquity is induced which acts to lift the leg vertically.

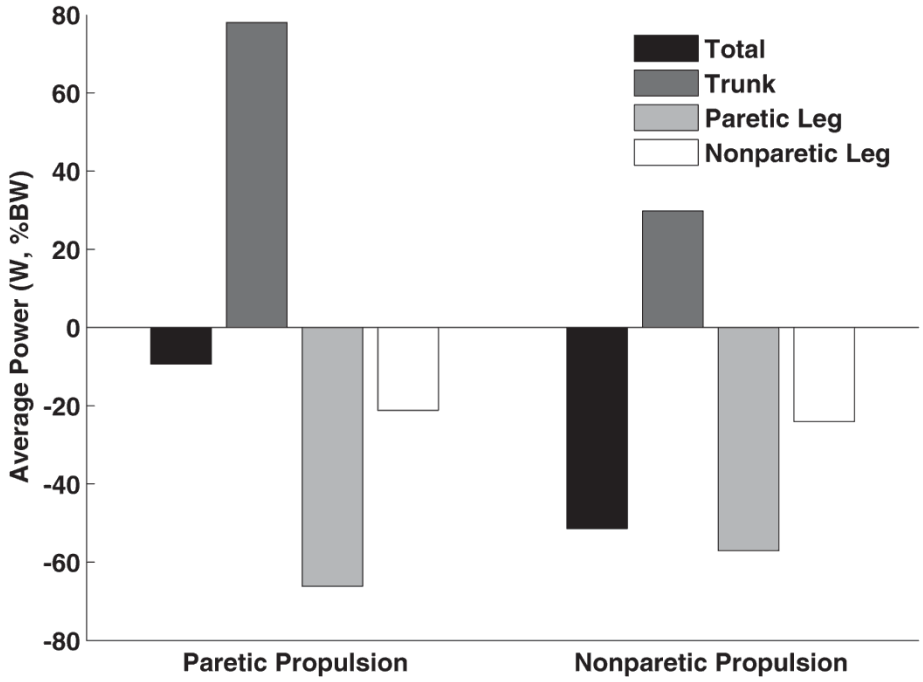

Figure 7: The paretic RF contributed positively to forward propulsion during both the paretic and nonparetic propulsive phase by transferring power from the legs to the trunk and pelvis.

Swing Initiation

Consistent with previous studies (Neptune et al., 2001; Neptune et al., 2008), the biarticular plantarflexors (GAS) contributed to swing initiation; the contributions, however, were low. Instead, both subjects relied on the hip muscles to generate power to the leg to accelerate it forward in pre-swing. The ipsilateral hip flexors (e.g. IL and AL) were key contributors to swing initiation in both subjects. Similar to previously reported IL results (Neptune et al., 2004), the AL accelerated the hip into flexion (Fig. 8b) and redistributed power from the trunk and contralateral leg to the ipsilateral leg (Fig. 8a).

Previous 2D simulations studies examining swing initiation do not include the AL muscles. AL was likely utilized in addition to IL in the current study because it induces non-sagittal plane hip motion (Fig. 8b), which is necessary for simulation of hemiparetic subjects who typically exhibit exaggerated pelvic hiking and limb circumduction (Chen et al., 2003; Chen et al., 2005; Lamontagne et al., 2007).

In addition to the hip flexors, the contralateral hip muscles also contributed highly to swing initiation. This is in agreement with a hip strategy proposed by McGibbon (2004), in which the hip extensor muscles of the stance leg act to tilt the pelvis posteriorly and help initiate swing of the contralateral leg (Fig 9b). For both subjects, the nonparetic anterior GMED was important for paretic swing initiation, generating power to the paretic leg through pelvis motion (Fig 9). Both subjects also utilized the paretic AM for nonparetic swing initiation in a similar manner. Both of these contributions were higher in the "hip strategy" subject.

HAM was also a key contributor to swing initiation. The nonparetic HAM was a major contributor for nonparetic swing initiation in both subjects. While the hamstrings are not active during pre-swing in healthy walking (Liu et al., 2006; McGowan et al., 2008; Neptune et al., 2004), some hemiparetic subjects exhibit prolonged nonparetic HAM activity, although typically only into single support (Den Otter et al., 2007). Hamstring activity during pre-swing is likely a similar strategy that both of these subjects used, which is seen in the subject's EMG patterns (Figs 10-13) and similar to the activity seen in a previous hemiparetic simulation study (Higginson et al., 2006). HAM contributes to swing initiation by inducing pelvic rotations that act to accelerate the leg in the direction of swing while also decelerating the pelvis COM (Fig. 6b).

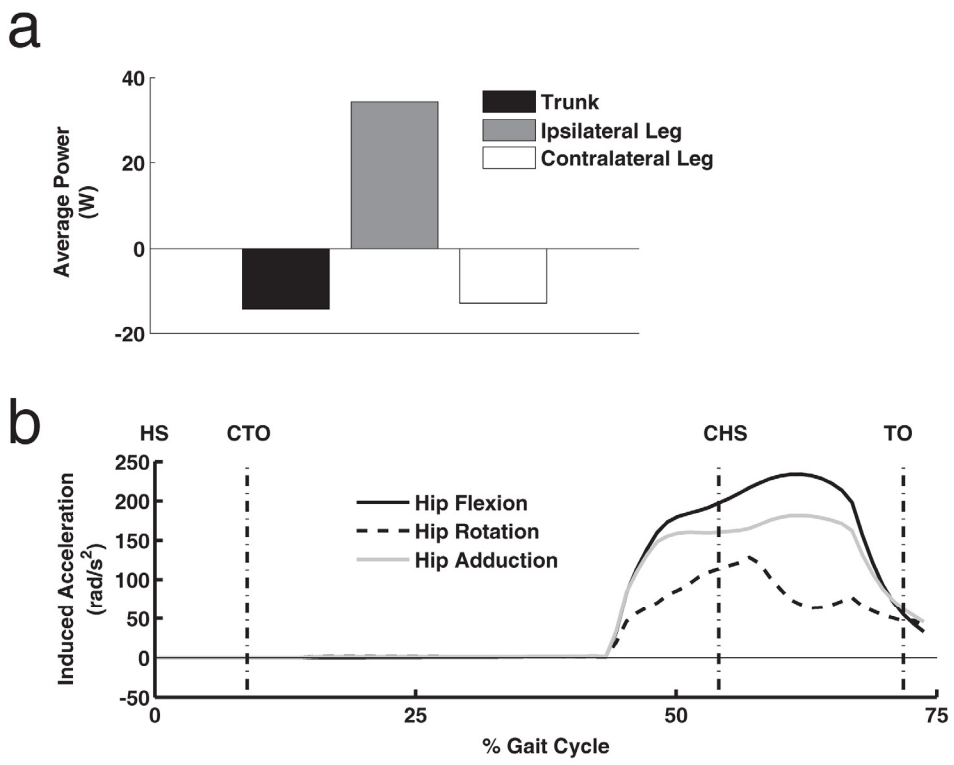

Figure 8: The ipsilateral AL (a) redistributed power from the trunk and contralateral leg to the ipsilateral leg during pre-swing and (b) accelerated the hip into flexion while also inducing non-sagittal rotations that are important in hemiparetic walking. HS – ipsilateral heel strike, CTO – contralateral toe off, CHS – contralateral heel strike, TO – ipsilateral toe off.

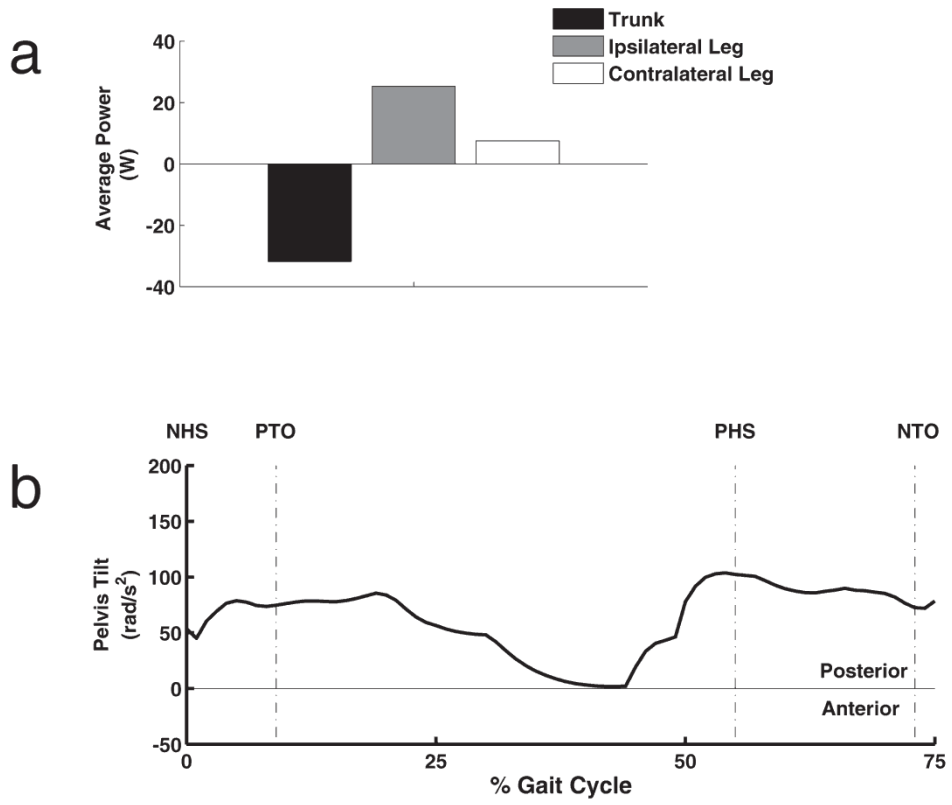

Figure 9: The nonparetic anterior GMED during paretic pre-swing (NHS to PTO) (a) generated power to both the nonparetic and paretic leg and (b) induced a posterior pelvis tilt. NHS – nonparetic heel strike, PTO – paretic toe off, NHS – nonparetic heel strike, PTO – paretic toe off.

Concluding remarks

In general, post-training muscle function was similar between subjects, with the level of individual muscle contributions varying between strategies. As expected, the plantarflexors (SOL, GAS) and hip flexors (IL) were important for forward propulsion and swing initiation, respectively. The inclusion of additional degrees of freedom at the pelvis, trunk, and hip in both the transverse and frontal planes revealed novel muscle function contributions to these walking subtasks; AL contributed to swing initiation and

the contralateral knee extensors (VAS and RF) contributed to forward propulsion. In addition, both subjects utilized the nonparetic HAM as a similar compensatory mechanism to help initiate swing in the nonparetic leg. There were very little differences between subjects in the level of contributions from the primary contributors to forward propulsion (contralateral VAS and ipsilateral SOL). This is consistent with the similar values of propulsion generated in the subjects post-training (Table 2). The main difference between the two strategies occurred primarily in the hip muscles for swing initiation. The "hip strategy" subject, in addition to using the hip flexors to accelerate the leg forward, had higher contributions from the contralateral non-sagittal plane hip muscles to generate energy to the leg to initiate swing.

Some of the important results of this study, such as the high contribution of the knee extensors (nonparetic VAS and paretic RF) to paretic forward propulsion in the "ankle strategy" subject, seem contrary to the subject's strategy classification. However, strategy classification was based on inverse dynamics results and thus focused on whole joint measures while muscle function post-training was determined using muscle-driven forward dynamics simulations. These results would not be evident from inverse dynamics alone and they highlight the benefit of using forward dynamics simulations to elucidate individual muscle function, showing that muscles not crossing the joints where the largest joint moment impulse changes occurred after training (Fig 2) are important for successful walking. The results of this study reveal that both the "ankle strategy" and the "hips strategy" utilize similar muscle functional output with differences primarily occurring in the hip muscle contribution to swing initiation. Future studies analyzing both pre- and post-training may reveal changes in muscle function that resulted from the locomotor strategy.

A limitation specific to this study is that model parameters were based on quantities found in healthy individuals while muscle weakness, spasticity, and joint stiffness, common among post-stroke hemiparetic individuals (Olney and Richards, 1996), were not included. For example, stroke survivors often have shorter optimal fiber length and increased muscle stiffness (Gao et al., 2009) that alters the intrinsic force-length relationship. However, changes in most of the muscle parameters related to these impairments would likely be compensated for by an increase or decrease in excitation magnitude such that the total force generated remains unaffected, provided that the excitation is sub-maximal. However, the simulation excitation patterns show several muscles near maximum activation (Figs. 10-13) and increases in excitation would not be possible for these muscles. In addition, some impairments post-stroke, such as increased joint stiffness, may alter muscle function. Further study is warranted to determine the effects of these impairments on muscle function.

An interesting question remains as to what predisposes a subject from using one strategy versus another. Many studies have reported an increase in metabolic cost in hemiparetic populations compared to healthy walkers (Bard, 1963; Detrembleur et al., 2003; Zamparo et al., 1995). One reason individuals may utilize different strategies is to minimize this cost, which likely depends on an individual's specific impairments and the remaining neuromuscular resources. In addition, rehabilitation may not be able to influence all impairments (e.g. contractures, disrupted neural pathways) that affect the resources on which an individual can draw when improving walking through locomotor-training. Because the effects of stroke are highly variable, the differences in functional resources available and the types of impairments may dictate the strategy used to increase speed throughout rehabilitation. Future research will include identifying and relating impairments pre-training to the strategy used to increase speed post-training, changes in

pre- and post-training muscle function, and relating strategy and impairments to variables that are easily measured by clinicians. Such an analysis will allow clinicians to develop rehabilitation training programs that are best suited for an individual's specific impairments.

Appendix

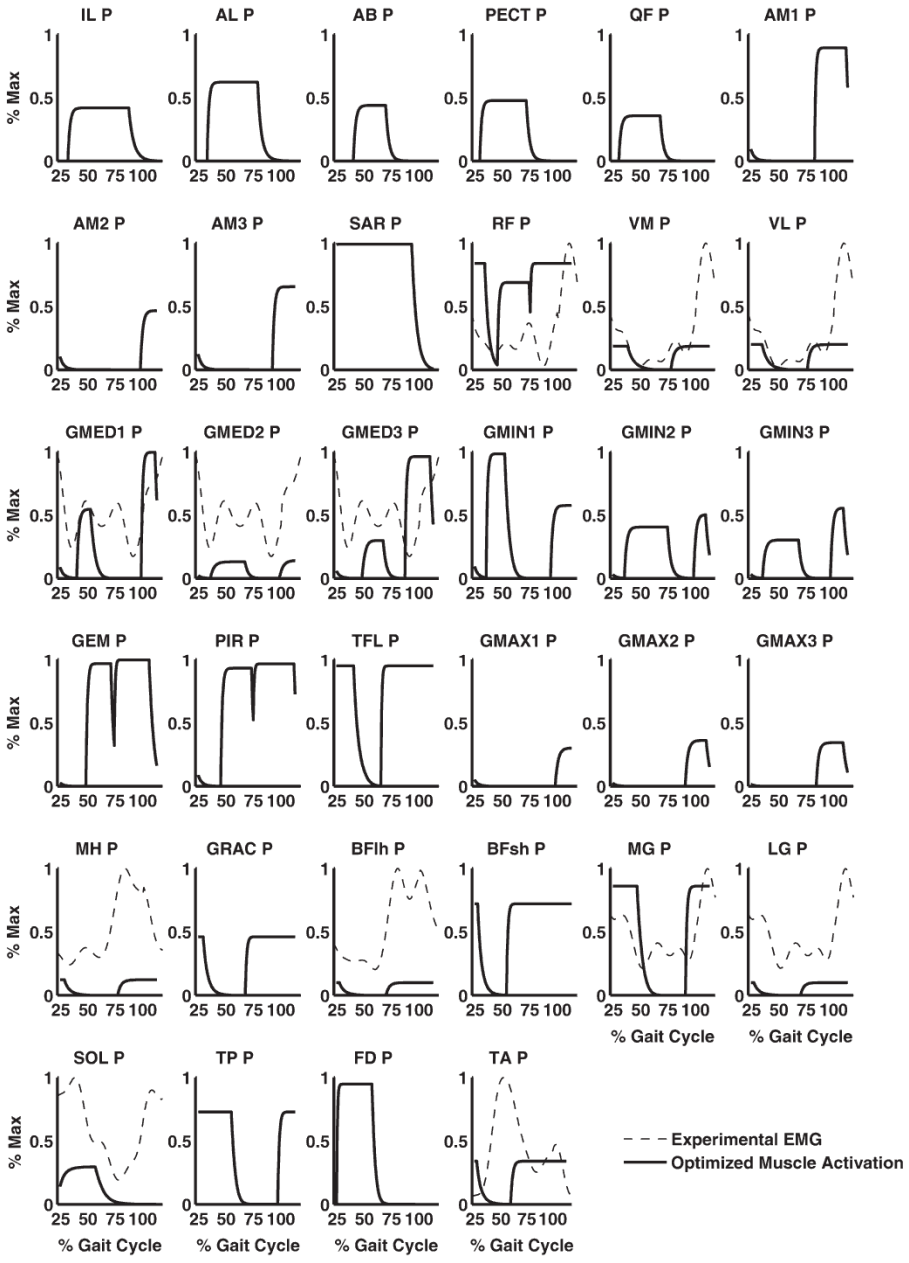

Figure 10: Subject 1 paretic leg simulation muscle activation and experimentally recorded EMG.

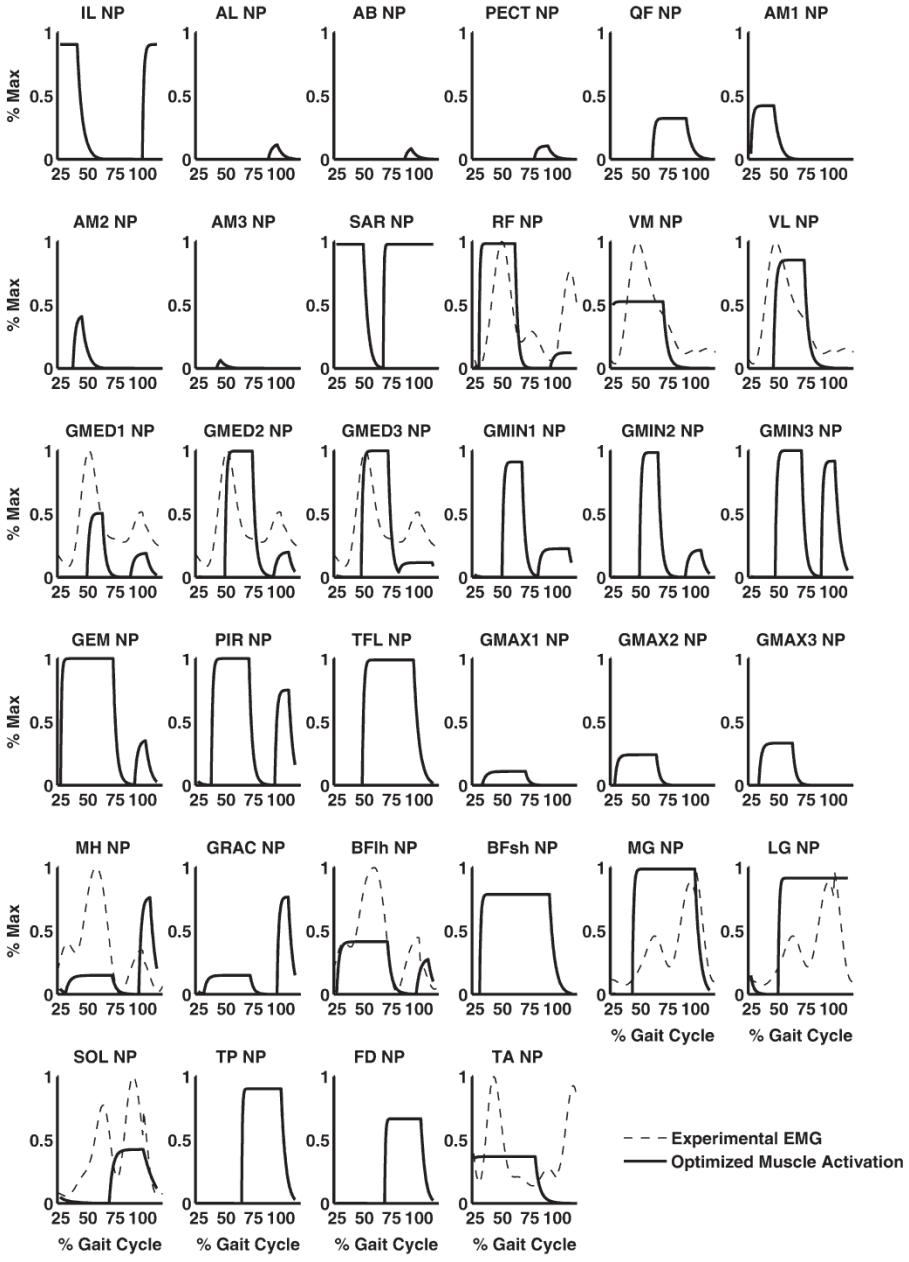

Figure 11: Subject 1 nonparetic leg simulation muscle activation and experimentally recorded EMG.

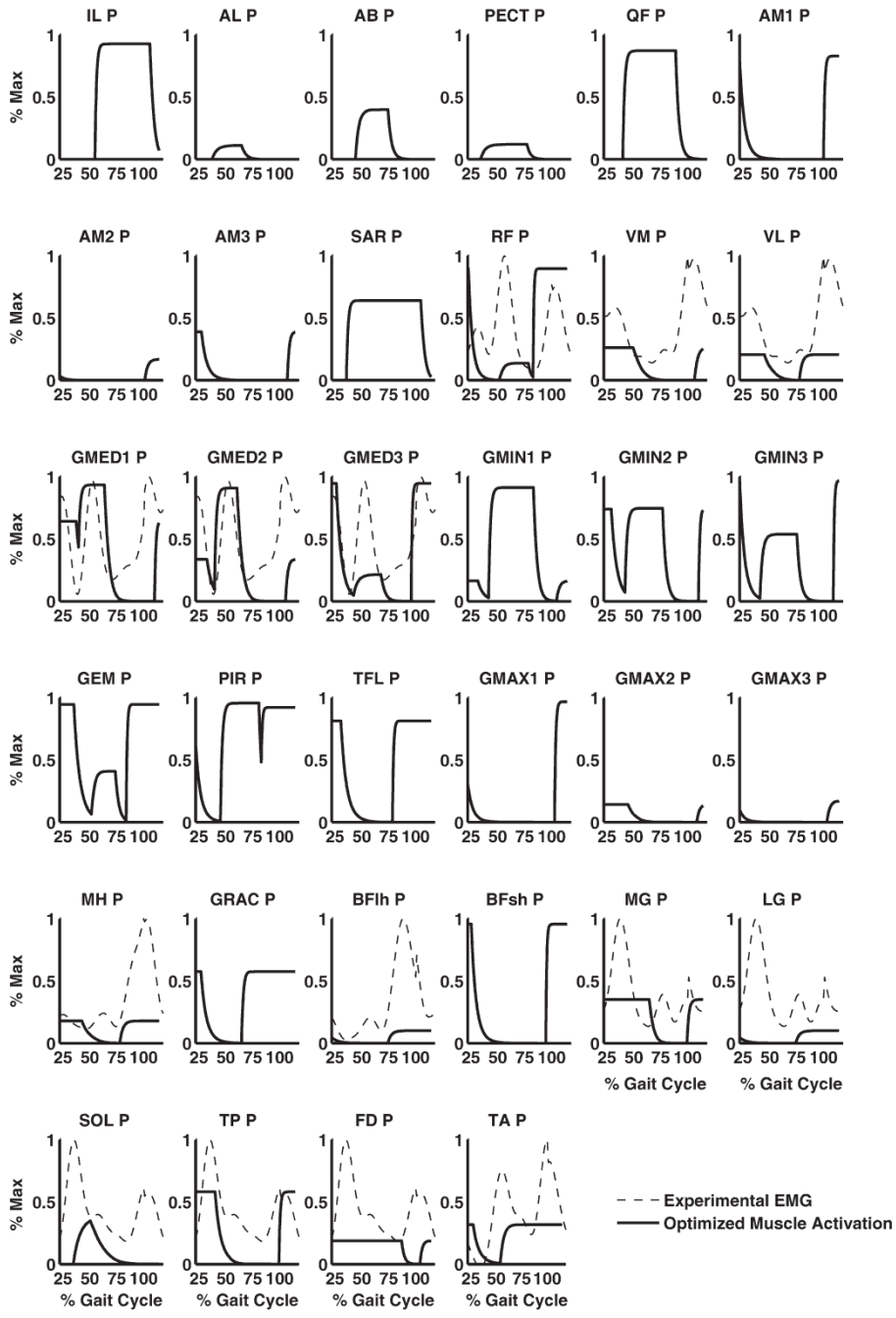

Figure 12: Subject 2 paretic leg simulation muscle activation and experimentally recorded EMG

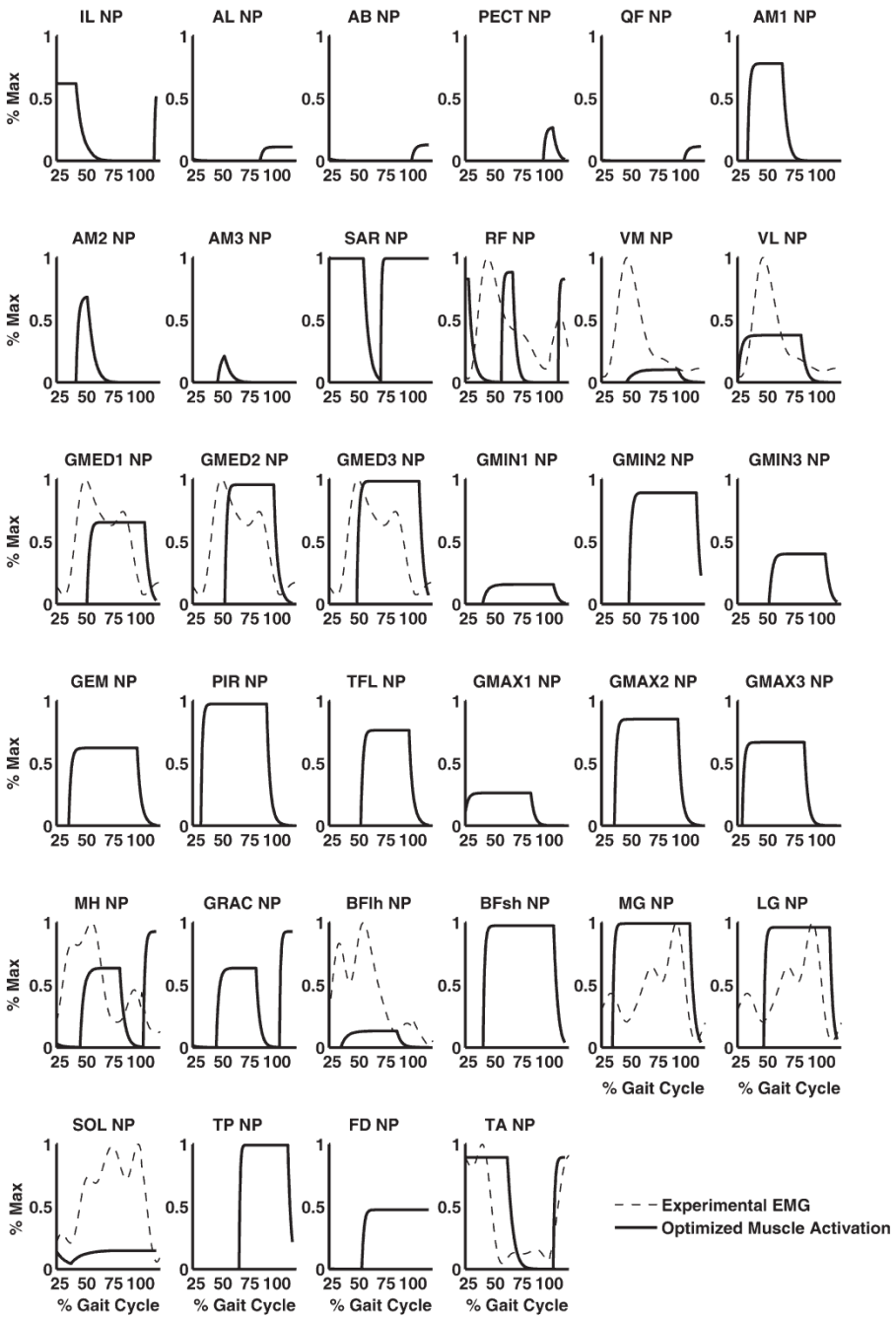

Figure 13: Subject 2 nonparetic leg simulation muscle activation and experimentally recorded EMG

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